AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph found on page 11, line 25 - page 12, line 14 with the following replacement paragraph.

Domain swapping between an avirulent 2b and its virulent homologue suggests a two-domain structure for an Avr protein. The N-terminal region of 69 amino acids of Tav2b, for example, constitutes the first domain that is essential and sufficient for resistance activation. Thus, both TMV-TC1 and TMV-TC2, encoding a complete resistance domain, induced strong virus resistance in the challenged tobacco plants. Complete (e. g., TMV-CT1,2 and 3) or partial (e. g., TMV-TC3) replacements or deletion (e. g.,TMV-TA2bl) of this domain result in the loss of the activity in resistance activation. The second domain, encompassing amino acids ~~70-9~~ 70-91 of Tav2b, is tentatively referred to as the "cell death"domain because its absence in TMV-TC2 leads to a loss of cell death induction, but without apparent effect on resistance activation. However, although the whole Cmv2b is inactive, it cannot be ruled out that the Cmv2b C-terminal 34 aa fused in TC2 may function as a positive or negative modulator of cell death initiation, leading to a local symptomless phenotype of TMV-TC2. Notably, these two functional domains correspond to the overlapping and non-overlapping regions of the cucumoviral 2b genes, as defined previously according to whether or not it overlaps with the 2a gene (Ding et al.,1995).

Please replace the paragraph found on page 12, line 34 - page 13, line 23 with the following replacement paragraph.

It will be of practical importance to determine if the concept of the two-domain structure of Tav2b also applies to Avr genes encoded by any other viral, bacterial and fungal pathogens. Constitutive expression of an Avr gene in a cultivar that contains the matching R gene should

generate a constitutivebroad-spectrum disease resistance. However, this type of resistance cannot be readily utilized because the specific Avr-R interaction also leads to immediate activation of the hypersensitive cell death (Culver and Dawson,1991; Gopalan et al., 1996; Leister et al., 1996; Scofield et al.,1996; Tang et al., 1996; Van denAckerveken et al., 1996; Gilbert et al., 1998). By removing the active cell death domain, swapping it with ~~ab~~ an inactive cell death domain, or selectively deactivating the cell death domain ~~will~~ while maintaining its macrostructural integrity, it would be possible to induce system resistance ~~with~~ without cell death. Because the SAR, once induced, is non-specific as to pathogen (be it viral, fungal, bacterial, etc.), it would be possible to confer broad-spectrum resistance by incorporating a single disarmed Avr(preferably disarmed by domain swapping) as a transgene. This is an efficient means of providing a pathogen-resistance plant.